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REPLY

Reply to A. D. Flouris and S. S. Cheung reply letter regarding "cold-induced vasodilation"

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Flouris and Cheung (2009a) contributed to the discussion on the methodology for inducing cold-induced vasodilation (CIVD) and concluded that CIVD is a centrally originating phenomenon caused by sympathetic vasoconstrictor withdrawal. In this reply we hope to show, using the data of the recent study they refer to (Flouris and Cheung 2009b), that CIVD is of peripheral origin.

First of all, there is no disagreement that CIVD is strongly influenced by the thermal state of the body. This is true when body heat content is modified by the temperature of drinks (Daanen et al. 1997) and also when the body is cooled or warmed from the outside (Daanen and Ducharme 1999).

When the body is thermoneutral to slightly cold, the finger blood flow is generally low. When a finger is immersed in cold water (<15°C) or cold air (<0°C, dependent on wind speed), the initial response is a strong vasoconstriction due to the reflexes initiated by the cold sensors in the fingers. After about 5–10 min the paradoxical CIVD response is initiated. It is paradoxical since the body is slightly cold, the hand is very cold and still large amounts of heat are released through the extremities (generally over 30 W for one hand). The mechanisms for this response are discussed previously and, in brief, the neuromuscular junction between the sympathetic nerve and muscular wall of the arterio-venous anastomosis (AVAs) is thought to be blocked by the local cold (Daanen 2003) leading to a strong increase in peripheral blood flow.

When the body is hypothermic, it is difficult to observe any CIVD response. Even though the AVAs open up, the arterioles leading to the AVAs are vasoconstricted, and consequently, the increase in peripheral blood flow is negligible. When the body is hyperthermic, there is a continuous heat loss from the fingers when immersed in cold water. This was recently confirmed in the elegant recent study of Flouris and Cheung (2009b), who investigated the effect of body temperature on CIVD while warming and cooling the body twice. The hand was exposed to 0°C air. The finger blood flow increased substantially during body warming. During the whole-body cooling period tympanic temperature and the body heat content dropped continuously. During the cooling process, there is a period during which the body core is in the range of thermoneutrality. During this period, the finger blood flow increased. This is shown in Fig. 1. The finger blood flow data is derived directly from Fig. 1 of the Flouris and Cheung paper (Flouris and Cheung 2009b); the body heat content was calculated from the Δ body heat content in Fig. 1 and follows a similar path to the tympanic temperature. Flouris and Cheung (2009b) claim that the subsequent increase in finger blood flow must be attributable to sympathetic vasoconstrictor withdrawal. However, this is not visible in their data. Indeed, as sympathetic activity is negatively correlated to core temperature (Sawasaki et al. 2001), one can conclude that the sympathetic activity must have been continuously increasing during the cooling period since tympanic temperature and body heat content was continuously decreasing. Yet, despite this continuous increase in sympathetic activity, a sudden increase in finger blood flow occurs. The obvious

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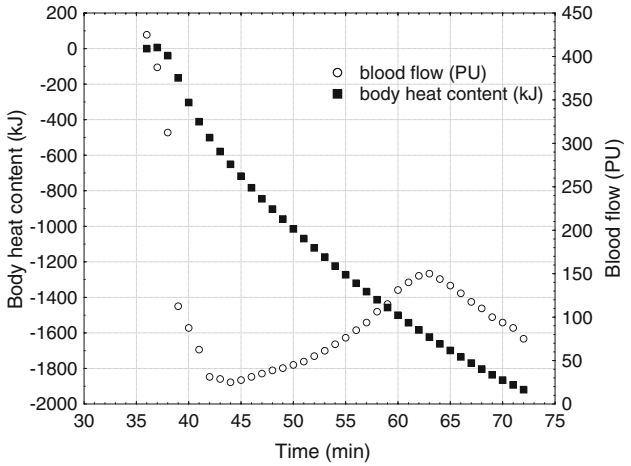

Fig. 1 Body heat content in kJ and finger blood flow in perfusion Units when the body is cooled in 12°C water and the hand is exposed to 0°C air (modified from Flouris and Cheung (2009b))

explanation is that CIVD of the fingers occurs and that the trigger to increase blood flow in the fingers was of peripheral rather than central origin. In Fig. 1 of the Flouris and Cheung paper (Flouris and Cheung 2009b) the finger skin temperature is continuously dropping. Thus, in line with previous observations summarized in Daanen (2003), the temperature of the AVAs may have become too low for effective neurotransmission at the junction of the sympathetic nerve and AVA, leading to a dilatation of the AVA and concomitant increase in blood flow and heat transfer.

The claim of Flouris and Cheung (2009a) that CIVD has been induced using whole-body cold exposure by several authors does not seem justified. Indeed, a careful reading of the literature showed that this is the case. Montgomery and Williams (1977) investigated heat loss, but not CIVD. Also, Berry et al. (1984) studied rat tail blood flow independently of ambient temperature, but not CIVD. Steegmann (1979) and Brajkovic and Ducharme (2006) exposed only the face to cold. Shitzer and his group modeled the response of fingers to local cold, and compared that to measurements of the fingers in 5°C water, but did not include whole-body cold exposure (Shitzer et al. 1996, 1997, 1998); Shitzer 1998. Flouris et al. (2008) observed fluctuations in finger skin temperature during whole-body exposure to cold air of sufficiently dressed subjects. The fluctuations were interpreted as cold-induced vasodilation (CIVD) by Flouris et al. (2008) and as a normal heat loss mechanism by Daanen (2009). Daanen (2009) argued that CIVD can only be evoked when the temperature of the extremity is below a certain threshold. Since the initial work of Lewis (1930), this upper limit in finger skin temperature is generally considered to be around 15°C, while the finger skin temperatures in the study of Flouris et al. (2008) were up to 33.5°C.

In summary, there is no indication that sympathetic withdrawal occurs when the body is exposed to cold. Therefore, the CIVD that is observed during cooling of the body has to be of peripheral origin, which is in line with the previously described temperature-dependent blockade of the neuromuscular junction at the arterio-venous anastomoses.

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